

[PubMed](#)[Nucleotide](#)[Protein](#)[Genome](#)[Structure](#)[PMC](#)[Taxonomy](#)[OMIM](#)[Bc](#)Search 

for

[Limits](#)[Preview/Index](#)[History](#)[Clipboard](#)[Details](#)[About Entrez](#)

Show:

[Text Version](#)[Entrez PubMed](#)[Overview](#)[Help | FAQ](#)[Tutorial](#)[New/Noteworthy](#)[E-Utilities](#)[PubMed Services](#)[Journals Database](#)[MeSH Database](#)[Single Citation Matcher](#)[Batch Citation Matcher](#)[Clinical Queries](#)[LinkOut](#)[Cubby](#)[Related Resources](#)[Order Documents](#)[NLM Gateway](#)[TOXNET](#)[Consumer Health](#)[Clinical Alerts](#)[ClinicalTrials.gov](#)[PubMed Central](#)[Privacy Policy](#)☐ 1: EMBO J. 1995 Nov 15;14(22):5589-96.[Related Articles, L](#)

## **A conserved domain in Bak, distinct from BH1 and BH2, mediates cell death and protein binding functions.**

**Chittenden T, Flemington C, Houghton AB, Ebb RG, Gallo GJ, Elangovan B, Chinnadurai G, Lutz RJ.**

Apoptosis Technology, Inc., Cambridge, MA 02139, USA.

Regulation of the cell death program involves physical interactions between different members of the Bcl-2 family that either promote or suppress apoptosis. The Bcl-2 homolog, Bak, promotes apoptosis and binds anti-apoptotic family members including Bcl-2 and Bcl-xL. We have identified a domain in Bak that is both necessary and sufficient for cytotoxic activity and binding to Bcl-xL. Sequences similar to this domain were identified in Bax and Bip1, two other proteins that promote apoptosis and interact with Bcl-xL, and were likewise critical for their capacity to kill cells and bind Bcl-xL. Thus, this domain is of central importance in mediating the function of multiple cell death-regulatory proteins that interact with Bcl-2 family members.

PMID: 8521816 [PubMed - indexed for MEDLINE]



Show:

[Write to the Help Desk](#)[NCBI | NLM | NIH](#)[Department of Health & Human Services](#)[Freedom of Information Act | Disclaimer](#)

Jul 17 2003 11:4